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Avian Influenza in North and South America, 2002–2005

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SUMMARY. Between 2002 and 2005, three outbreaks of highly pathogenic avian influenza (HPAI) occurred in the Americas: one outbreak in Chile (H7N3) in 2002, one outbreak in the United States (H5N2) in 2004, and one outbreak in Canada (H7N3) in 2004. The outbreak in Chile was limited to a large broiler breeder operation and a nearby turkey flock and represented the first outbreak of HPAI in that country. The outbreak of HPAI in the United States occurred in Texas and was limited to one premise where chickens were raised for sale in nearby live-bird markets. The outbreak in Canada was the largest of the three HPAI outbreaks, involving 42 premises and approximately 17 million birds in the Fraser Valley, British Columbia. In each of the HPAI outbreaks, the disease was successfully eradicated by depopulation of infected farms. All other reports of infections in poultry and isolations from wild bird species pertained to low pathogenicity avian influenza (LPAI) viruses. Animal Health Officials in Canada reported subtypes H3, H5, and H6 in domestic poultry, and H3, H5, H11, and H13 from imported and/or wild bird species. An LPAI H5N2 virus continues to circulate in Mexico and the Central American countries of Guatemala and El Salvador. Each country reported isolations of H5N2 virus from poultry and the large-scale use of inactivated and recombinant H5 vaccines in their AI control programs. In Colombia, AI was reported for the first time when antibodies to H9N2 were detected in chickens by routine surveillance. Intensive surveillance activities in the United States detected AI virus or specific antibodies to 13 of the 16 hemagglutinin (H1–H13) and all nine neuraminidase subtypes in live-bird markets, small holder farms, and in commercial poultry from 29 states. The largest outbreak of LPAI in the United States occurred in 2002, when 197 farms were depopulated (4.7 million birds) to control an outbreak in Virginia and surrounding states. The outbreak was caused by an LPAI H7N2 virus closely related to an H7N2 virus that has been circulating in the live-bird marketing system in the northeastern United States since 1994.

RESUMEN. Influenza aviar en Norte y Suramérica, 2002–2005

Entre los años 2002 y 2005, han ocurrido tres brotes de virus de alta patogenicidad de influenza aviar en el continente Americano: Un brote en Chile (H7N3) en el año 2002, uno en Estados Unidos (H5N2) en el 2004 y uno en Canadá (H7N3) en este mismo año. El brote en Chile se limitó a una gran explotación de reproductoras pesadas y a un lote cercano de pavos y representó el primer brote de virus de influenza de alta patogenicidad en ese país. El brote de influenza de alta patogenicidad en los Estados Unidos ocurrió en Texas y se limitó a un local donde los pollos eran criados para venderlos en mercados cercanos de aves vivas. El brote en Canadá fue el mayor de los tres, involucrando 42 explotaciones y aproximadamente 17 millones de aves en el valle de Fraser, Columbia Británica. En cada uno de los brotes de influenza aviar de alta patogenicidad, la enfermedad fue erradicada con éxito mediante la despoblación de granjas afectadas. Todos los demás reportes de infección en aves comerciales y los aislamientos de aves silvestres pertenecieron a virus de influenza aviar de baja patogenicidad. Los funcionarios de Sanidad Animal en Canadá reportaron la presencia de los subtipos H3, H5 y H6 en aves domésticas, y los subtipos H3, H5, H11 y H13 de aves importadas y en especies silvestres. Un virus de baja patogenicidad H5N2 continúa circulando en México y en los países centroamericanos de Guatemala y El Salvador. Cada país reportó el aislamiento de virus H5N2 de aves domésticas y el uso a gran escala de vacunas H5 inactivadas y recombinantes en sus programas de control de la Influenza aviar. En Colombia la influenza aviar fue reportada por primera vez cuando se detectaron anticuerpos para virus H9N2 en pollos durante el desarrollo de vigilancia epidemiológica de rutina. En Estados Unidos las intensas actividades de vigilancia detectaron el virus de Influenza aviar o anticuerpos específicos para 13 de 16 hemoaglutininas (H1–H13) y en todos los nueve subtipos de neuraminidasa en mercados de aves vivas, granjas pequeñas y en aves comerciales de 29 estados. El mayor brote de influenza aviar de baja patogenicidad en los Estados Unidos ocurrió en el año 2002, cuando 197 granjas fueron despobladas (4.7 millones de aves) para controlar el brote en el estado de Virginia y los estados adyacentes. El brote fue causado por un virus de baja patiogenicidad H7N2 que estaba relacionado estrechamente con un virus H7N2 que ha circulado en el sistema de mercados de aves vivas en el Noroeste de los Estados Unidos desde el año 1994.

Key words: avian influenza, highly pathogenic avian influenza, low pathogenicity avian influenza, live-bird markets

Abbreviations: AI = avian influenza; HA (H) = hemagglutinin; HPAI = highly pathogenic avian influenza; IVPI = intravenous pathogenicity index; LBM = live-bird market; LBMS = live-bird marketing system; LPAI = low pathogenicity avian influenza; NA (N) = neuraminidase; OIE = World Organisation for Animal Health

The following report is a brief account of avian influenza (AI) virus detections in domestic poultry and wild birds in North and South America from 2002 to 2005. Occurrences of highly pathogenic avian influenza (HPAI) outbreaks were obtained from reports to the World Organisation for Animal Health (OIE) or by direct involvement as an OIE reference laboratory. However, gathering information on the occurrences of low pathogenicity avian influenza (LPAI) is more difficult because there is no centralized source of surveillance information. Some countries in the region may have AI surveillance programs in place, but the information was not available for inclusion in this report. Reports of AI from the United States were obtained from the Proceedings of the U.S. Animal Health Association (12) or from personal observations. The information is reported by species.

HPAI IN CHICKENS

Between 2002 and 2005, there were three outbreaks of HPAI in chickens in the Americas. Each of the outbreaks was caused by an H5 or H7 AI virus that was shown to have mutated to HPAI from

an LPAI precursor virus after circulating in poultry for a period of 2 wk to 2 yr. Additionally, each of the three HPAI viruses responsible for the outbreaks had unique characteristics and fulfilled either the chicken virulence criterion or the molecular criterion for HPAI but not both (13). In two of the outbreaks (Chile, 2002 and Canada, 2004), homologous recombination was shown to be the mechanism by which the viruses made the switch from LPAI to HPAI, and they represent the first reports of recombination of genes with AI viruses under field conditions. Details of the outbreaks and the unique characteristics of the viruses are given below.

Chile. In early May, 2002 a broiler breeder company near Santiago observed a drop in egg production, respiratory signs, salpingo-peritonitis, and an increase in mortality in a multiage complex of approximately 600,000 birds. Infectious bronchitis was initially suspected as the cause of the disease. Specimens were collected and sent to a Ministry of Agriculture Laboratory in Santiago where a hemagglutinating virus was isolated. The virus was subsequently identified as H7N3 AI virus and was characterized as LPAI by the chicken pathogenicity test (intravenous pathogenicity index [IVPI] = 0.0) and by deducing the amino acid motif at the cleavage site of the hemagglutinin protein (PE**K**P**K**T**R**/GLF).

By late May 2002, the severity of the disease in the field quickly changed. The breeders experienced a precipitous drop in egg production with mortalities exceeding 100,000 birds. Many of the birds had gross lesions compatible with that of HPAI. Based on these observations, a decision was made by the Chilean Ministry of Agriculture to depopulate all chickens in the premises without a laboratory confirmation of HPAI. Samples collected at the time of depopulation yielded several H7N3 viruses that were subsequently characterized as HPAI viruses. The IVPIs of the viruses ranged from 2.4 to 3.0, and two amino acid motifs were observed: PE**K**P**K**TCSPLS**R**C**RK**T**R**/GLF and PE**K**P**K**TCSPLS**R**C**R**ET**R**/GLF. Each of the isolates had a 30-nucleotide insert (underlined) near the cleavage site of the HA as a result of recombination between the hemagglutinin (HA) and nucleoprotein genes of the LPAI virus (10). Sequence comparisons of all eight gene segments showed the Chilean viruses were distinct from all other H7 AI viruses and represent a distinct South American clade (10). The outbreak was limited to the index premises and a nearby complex of 50,000 turkey breeders. The turkey farm was depopulated by mid-June 2002.

United States. On February 23, 2004, HPAI H5N2 was confirmed in a flock of 6600 broilers near Gonzales, TX. This outbreak was the first outbreak of HPAI in the United States since the HPAI outbreak of H5N2 in 1983–84. The broilers were being raised for sale in live-bird markets (LBMs) in Houston, TX. A shipment of chickens to the LBMs had been made within a week of the outbreak. Testing in the Houston LBMs found two of the five markets positive for the H5N2 virus. Clinical signs in the source flock were consistent with LPAI and included respiratory signs (moist rales and gasping) with increased mortalities. The flock was also positive for *Mycoplasma galisepticum*. The virus was unique because it met the molecular criterion for HPAI based on presence of multiple basic amino acids at the cleavage site of the HA protein (PQ**RKKR**/GLF) but was not pathogenic (IVPI = 0.0) for chickens. The cleavage site sequence is the same for that of A/Chicken/Scotland/59, a known HPAI virus. The Texas/04 H5N2 virus was shown to have a similar HA gene sequence as an H5N3 AI virus (PQ**R**E**KR**/GLF) also isolated from chickens in Texas in 2002, but it had acquired one more basic amino acid at the cleavage site by a single point mutation (5). The isolate also shared similar sequence with the 2002 H5N3 virus in several internal genes, but the polymerase genes (PA, PB1, and PB2) had sequence of clearly different origin, which indicated the virus had undergone reassortment (5). The outbreak was limited to the index farm and the two LBMs. All five LBMs and the index farm were depopulated.

Canada. An outbreak of HPAI caused by H7N3 subtype was confirmed on March 8, 2004, on a farm in Abbotsford, British Columbia. This outbreak was the first outbreak of HPAI in Canada since 1966. The farm was a broiler breeder facility that supplied hatching eggs to a local hatchery. When the disease first occurred in early February, 2004, there were two houses of birds (about 9000 birds each) present on the farm, an older flock (52 weeks of age) and a younger flock (24 weeks of age). Clinical signs in the 52-week-old flock included a slight drop in egg production and a slight increase in mortality (six birds per day). Pathologic lesions were congested lungs and inflamed tracheas. An H7N3 AI virus characterized as LPAI was isolated from the 52-week-old birds, and the flock seemed to recover from the disease. However, on February 16, AI infection was detected in the younger flock (24 weeks of age) by polymerase chain reaction assay, and within a day a sharp rise in mortality (>900 birds) was reported. Subsequently, an HPAI H7N3 virus was isolated from the flock. The HPAI virus was unusual in that the amino acid sequence at the cleavage site of the HA protein (PENP**K**QAY**RKR**MT**R**/GLF) had an insert of 7 amino acids (underlined) derived from the matrix gene (2,4). This report is the second report (Chilean outbreak being the first report) of an insertion of nucleotides at the HA cleavage site by homologous recombination. Eventually, the virus spread to 42 premises, and a total of approximately 17 million birds were destroyed to control the outbreak. Additional details of the outbreak are provided by other articles in this symposium.

LPAI IN CHICKENS

AI virus infections in chickens were reported from Mexico, Guatemala, Colombia, and the United States. A list of the virus subtypes isolated is shown in Table 1, and list of specific antibodies to AI virus is shown in Table 2.

Mexico. The LPAI H5N2 virus that mutated to cause the outbreak of HPAI in Mexico in 1994–95 continues to circulate in chickens in central Mexico. The LPAI H5N2 virus was isolated each year from 2002 to 2005, with the number of isolates per year ranging from nine to 63, and the number of positive states ranging from three to seven. Control efforts have centered on the use of vaccine. During 2002–05, approximately 558.3 million doses of vaccine were used; 364.3 million doses of inactivated, whole virus H5 vaccine and 194 million doses of a recombinant fowlpox-H5 vaccine. No other subtypes of AI virus were reported.

Central America. An LPAI H5N2 virus was first detected in the Central American countries of Guatemala and El Salvador in 2000 and 2001, respectively. The virus was shown to be genetically related to the H5N2 virus that has circulated in Mexico since 1994. As in Mexico, the control efforts have centered on vaccination. From both countries, reports of sporadic isolations of the H5N2 virus have been made since 2002. Surveillance for AI is being conducted in Belize, Costa Rica, Honduras, and Nicaragua but there have been no reports of infection in those countries.

South America. Avian influenza was detected for the first time in Colombia when antibodies to H9N2 were detected in a flock of broiler breeders as a result of active surveillance for AI. No clinical signs were reported in the flock, and attempts to isolate the virus were unsuccessful. There were no other reports of AI infections in chickens in South America.

United States. Active and passive surveillance programs for AI infections in chickens in the United States detected nine HA (H1, H2, H3, H4, H5, H6, H7, H9, and H10) and six neuraminidase

Table 1. Subtypes of AI virus isolated from North and South America from 2002 to 2005. All viruses were characterized as low pathogenicity viruses unless otherwise indicated. Postal codes are used for states or provinces.

Subtype	Host(s)	Country (state/province)	Yr
H1N1	Unknown avian	U.S.A. (NY)	2005
H1N4	Duck	U.S.A. (ME)	2002
H2N2	Chicken	U.S.A. (NJ, NY, PA)	2004
H2N3	Unknown avian	U.S.A. (NY)	2005
H3N2	Duck	U.S.A. (NY)	2002
	Duck	U.S.A. (CA)	2003
	Turkey	U.S.A. (NC)	2003
	Turkey	U.S.A. (NY, OH)	2004
	Turkey	Canada (BC, MB, ON)	2005
H3N5	Duck	U.S.A. (NJ)	2002
H3N6	Chicken	U.S.A. (NY)	2002
	Duck	U.S.A. (NY)	2004
H3N8	Chicken	U.S.A. (NY)	2002, 2003
	Pet bird (imported)	Canada	2003
H3N9	Duck	U.S.A. (ME)	2002
H4N1	Duck	U.S.A. (NC)	2002
H4N2	Chicken	U.S.A. (NY, PA)	2005
	Turkey	U.S.A. (NC)	2003
H4N6	Duck	U.S.A. (IN, NY)	2002
	Duck	U.S.A. (CA, NY)	2003
	Duck	U.S.A. (PA)	2004
	Duck	U.S.A. (MA)	2005
	Guinea fowl	U.S.A. (RI)	2002
	Unknown avian	U.S.A. (NJ)	2005
H4N8	Turkey	U.S.A. (CA)	2005
H5N1[A]	Wild duck	Canada (MB, ON)	2005
H5N2[B]	Chicken	U.S.A. (TX)	2004
H5N2	Chicken	Guatemala	2002, 2003
	Turkey	U.S.A. (CA)	2002
	Chicken	Mexico	2002, 2003, 2004, 2005
	Chicken	U.S.A. (NY)	2002
	Duck	U.S.A. (ME, NY)	2002
	Duck	U.S.A. (SC)	2004
	Amazon parrot	U.S.A. (CA)	2002
	Unknown avian	U.S.A. (NY)	2005
	Duck	U.S.A. (NY)	2005
	Wild duck	Canada (BC)	2005
H5N3	Chicken	U.S.A. (TX)	2002
	Wild duck	Canada (QC)	2005
H5N4	Duck	U.S.A. (NY)	2002
H5N5	Duck	U.S.A. (MA), Canada (BC)	2005
H5N8	Duck	U.S.A. (NY)	2002
	Environment	U.S.A. (NY)	2003
H5N9	Duck	U.S.A. (NY)	2003
	Wild duck	Canada (BC)	2005
H6N2	Duck	U.S.A. (NY)	2002, 2003, 2004
	Chicken	U.S.A. (CA)	2002, 2003, 2004
	Duck	U.S.A. (PA)	2003
	Turkey	U.S.A. (CA)	2002, 2003
	Turkey	U.S.A. (NY)	2003
	Quail	U.S.A. (CA)	2004
H6N6	Duck	U.S.A. (NY)	2002
	Chicken	U.S.A. (CA)	2004
H6N8	Waterfowl	U.S.A. (GA)	2002
	Guinea fowl	U.S.A. (PA)	2005
	Turkey	Canada (ON)	2003
H7N2	Chicken	U.S.A. (CT, MA, NC, PA, VA, WV)	2002
	Turkey	U.S.A. (NC, VA)	2002
	Duck	U.S.A. (PA)	2002
	Chicken	U.S.A. (CT)	2003
	Chicken	U.S.A. (NY)	2002, 2003, 2004
	Chicken	U.S.A. (DE, MD)	2004
	Duck	U.S.A. (NY)	2004
	Chicken	U.S.A. (NJ)	2002, 2004, 2005
	Chicken, duck	U.S.A. (NY)	2005
H7N3[B]	Chicken	Chile, LPAI and HPAI	2002
H7N3[B]	Chicken	Canada (BC), LPAI and HPAI	2004
H7N3	Duck	U.S.A. (PA)	2002
	Chicken	U.S.A. (MA)	2004
	Unknown avian	U.S.A. (NY)	2004
H8N4	Turkey	U.S.A. (CO)	2002, 2003
H9N2	Duck	U.S.A. (NY)	2002, 2003
	Unknown avian	U.S.A. (NY)	2004
H10N7	Unknown avian	U.S.A. (NJ)	2002
H10N9	Chicken	U.S.A. (FL)	2005
H11N3	Duck	U.S.A. (MD)	2005
H11N6	Duck	U.S.A. (NY)	2002
	Unknown avian	U.S.A. (NY)	2003
H11N9	Duck	U.S.A. (NY)	2002
	Duck	Canada (BC)	2004
H13N6	Gull	Canada (ON)	2002

[A]North American lineage.
[B]HPAI.

(NA) (N2, N3, N6, N7, N8, and N9) subtypes of AI virus and/or specific antibodies from 16 states (Tables 1, 2). Most of the infections were detected in LBMs and small-holder premises. However, there were several notable outbreaks of LPAI in commercial poultry.

In May 2002, an infection with LPAI H5N3 was diagnosed in two flocks of commercial chickens in Texas. Both farms were depopulated. The origin of this virus was not determined, but a link was established between the index farm and the LBMs in Houston. The H5N3 virus is thought to be the precursor to the HPAI H5N2 virus, isolated also in Texas in 2004 (see summary of HPAI outbreak above).

Also, in 2002 (September) LPAI H5N2 was isolated from a single grandparent flock of turkey breeders in California. No further spread of the virus was detected. The farm was voluntarily depopulated by the owner.

Antibodies to H5N1 were detected in serum samples collected at slaughter from a single flock of meat turkeys in Michigan in 2002. The infecting virus is assumed to have been a low pathogenicity H5N1 virus based on the lack of clinical signs in the flock and inspection of antemortem records. Surveillance of other flocks in the area was negative for avian influenza virus (AIV). This detection is the first and only detection of H5N1 AIV in commercial poultry the United States.

In early March 2003, a major outbreak of LPAI H7N2 occurred in several multi-age, in-line, table egg layer operations in Connecticut (CT). The outbreak involved four of seven farms owned by a single company and affected approximately 3.5 million layers and 1.2 million replacement pullets. Affected flocks experienced a respiratory disease and a 10–20% drop in egg production that lasted for about 2 wk before returning to near normal production levels. One additional layer flock of 30,000 in Rhode Island (RI) was also positive for the H7N2 virus. Studies on the CT and RI isolates showed the viruses were related to the H7N2

Table 2. AI virus subtype specific antibodies detected in serum from various sources in the United States and Colombia, 2002–2005. Postal codes are used for states.

Subtype	Host(s)	Country (state/province)	Yr
H1	Chicken	U.S.A. (CA, MD, WV)	2002
	Chicken	U.S.A. (IN, WA)	2004
	Quail	U.S.A. (CA)	2004
	Turkey	U.S.A. (MN)	2002
	Turkey	U.S.A. (MI)	2005
H1N1	Pigeon	U.S.A. (CA)	2003
	Turkey	U.S.A. (IA, IL, MN, NC, ND, OH, SC)	2002
	Turkey	U.S.A. (MN, NC, OH)	2003
	Turkey	U.S.A. (IA, MI, MN, NC, OH)	2004
	Turkey	U.S.A. (IA, IL, IN, MN, NC, OH, SD)	2005
H1N2	Turkey	U.S.A. (IA, ND)	2004
	Turkey	U.S.A. (OH)	2005
H2	Chicken	U.S.A. (WA)	2004
	Duck	U.S.A. (PA)	2002
H2N1	Turkey	U.S.A. (MN)	2004
H2N2	Chicken	U.S.A. (PA)	2004, 2005
	Loon	U.S.A. (ME)	2005
H2N3	Duck	U.S.A. (PA)	2005
H3	Chicken	U.S.A. (IN, SD)	2004
	Chicken	U.S.A. (AL, FL)	2005
	Pheasant	U.S.A. (TX)	2004
	Turkey	U.S.A. (MN)	2004
	Turkey	U.S.A. (MI)	2005
	Quail	U.S.A. (CA)	2004
H3N1	Turkey	U.S.A. (MN)	2005
H3N2	Chicken	U.S.A. (WA, WV)	2004
	Chicken	U.S.A. (IA, NY)	2005
	Duck	U.S.A. (CA)	2003
	Turkey	U.S.A. (IA, MI, MO, OH, SD)	2004
	Turkey	U.S.A. (IA, IN, MN, NC, OH, SD)	2005
H3N5	Duck	U.S.A. (NJ)	2002
H4	Chicken, duck	U.S.A. (PA)	2004
H4N2	Turkey	U.S.A. (MA)	2005
H4N6	Duck	U.S.A. (NY)	2003
	Turkey	U.S.A. (VA)	2005
	Duck	U.S.A. (PA)	2005
H4N8	Turkey	U.S.A. (CA)	2005
H5	Duck	U.S.A. (NJ)	2002
	Chicken	U.S.A. (NY)	2005
H5N1	Turkey	U.S.A. (MI)	2002
H5N2	Turkey	U.S.A. (CA)	2002
	Duck	U.S.A. (ME, NY)	2002
	Chicken	U.S.A. (DE, TX)	2004
	Duck	U.S.A. (SC)	2004
	Duck	U.S.A. (NY)	2005
H5N3	Duck	U.S.A. (CA)	2002
	Chicken	U.S.A. (TX)	2002
H6	Chukar partridge	U.S.A. (CA)	2004
	Chicken	U.S.A. (RI)	2005
H6N2	Chicken	U.S.A. (CA)	2002, 2003, 2005
	Goose	U.S.A. (VA)	2002
	Goose	U.S.A. (PA)	2005
	Turkey	U.S.A. (CA)	2002, 2003
	Quail	U.S.A. (CA)	2004
H6N5	Duck	U.S.A. (CA)	2002
H6N8	Duck	U.S.A. (PA)	2004
H7	Quail	U.S.A. (SC)	2002
H7N2	Chicken	U.S.A. (CT, NC, NY, VA, WV)	2002
	Chicken	U.S.A. (CT, RI)	2003
	Chicken	U.S.A. (NY, RI)	2004
	Duck	U.S.A. (NY)	2005
	Turkey, quail	U.S.A. (NC)	2002
	Turkey	U.S.A. (VA)	2002
H7N3	Chicken	U.S.A. (MA, TX)	2004
H8N4	Turkey	U.S.A. (CO)	2002, 2003
H9	Pheasant	U.S.A. (TX)	2004
	Chicken	U.S.A. (RI)	2005
H9N2	Duck	U.S.A. (CA)	2002
	Chicken	Colombia	2005
H9N9	Turkey	U.S.A. (MN)	2002
H10	Duck	U.S.A. (CA)	2005
H10N7	Turkey	U.S.A. (MN)	2002
	Chicken	U.S.A. (FL)	2005
H11	Duck	U.S.A. (MD, PA)	2005
H11N2	Pelican	U.S.A. (FL)	2002
H12	Turkey	U.S.A. (OH)	2005

virus that has been circulating in the live-bird marketing system (LBMS) in northeastern United States. There was no epidemiologic connection between the CT and RI outbreaks. The outbreak in CT was successfully controlled, and the LPAI H7N2 virus was eradicated from the flocks by the use of a comprehensive vaccination program. The vaccination program included two doses of vaccine for non-infected replacement pullets and a single dose of vaccine for birds previously infected with the LPAI H7N2 virus. In addition to the use of vaccine, the program included a review of the company's biosecurity program and an extensive monitoring for virus and antibodies in vaccinated and sentinel birds. The program also outlined procedures for the treatment, testing and disposal of manure, and disposition of birds at the end of production. Later, vaccinated birds were removed from the premises, and all birds tested have been negative for antibodies to the H7N2 AIV.

In 2003, LPAI H6N2 was isolated from several flocks of table-egg layers in California (CA). The H6N2 virus was first detected in CA in 2000 and again in 2002, 2003, and 2004. An autogenous, killed H6N2 vaccine was developed to control and eradicate the H6N2 virus from chickens in CA. There have been no reports of additional infections with H6N2 in commercial poultry since 2004.

In 2004, two flocks of chickens (84,000 birds) in the dense broiler production region in Delaware (DE) and one flock (112,000) in Maryland (MD) were infected with LPAI H7N2. The DE and MD outbreaks were not epidemiologically linked; however, the H7N2 virus from both outbreaks was shown to be closely related to the H7N2 virus circulating in the LBMS. The index flock of broilers in DE had delivered a shipment of birds to a LBM in New Jersey a few days before appearance of clinical disease. All three farms were depopulated.

In 2004, two table-egg layer flocks in Pennsylvania (PA) were infected with an LPAI H2N2 virus. The virus was shown to be of avian origin and likely from wild birds. Two additional layer flocks in PA, one flock in 2004 and one flock in 2005, were found to be positive for antibodies to the H2N2 virus. No clinical disease was observed in any of the flocks and serologic testing indicated a low prevalence of infection. The positive flocks were detected through routine National Poultry Improvement Plan surveillance testing. The flocks were kept under quarantine until shown to be virus negative and then sent for processing at the end of the lay cycle.

In May 2005, a LPAI H7N2 was isolated from a duck production facility in New York (NY). The facility was immediately quarantined. The state of NY and the flock owners are continuing to clean and disinfect between duck placements while sending ducks to an on-site USDA processing facility.

In 2005, an LPAI H4N2 was isolated from a chicken flock in PA. No clinical disease was reported.

LPAI IN TURKEYS

Canada. The only reports of LPAI infections in turkeys in the Americas came from Canada and the United States. In 2003, Canadian Animal Health Officials reported a single case of H6N8 in a commercial meat-turkey farm in Ontario. Clinical signs included a respiratory disease with airsacculitis, pneumonia, and increased mortality. In 2005, three turkey breeder flocks in British Columbia were infected with H3N2 subtype virus and in 2006 a single turkey breeder flock in Manitoba was also positive for the H3N2 virus. Clinical signs in the turkey breeders infected with the H3N2 virus were limited to a drop in egg production. In all four flocks, the H3N2 virus was shown to be closely related to the H3N2 virus currently circulating in swine in North America (Tables 1, 2) (Pasick, pers. comm.).

United States. Surveillance for AI infections in turkeys in the United States detected virus or antibodies to 11 HA (H1, H2, H3, H4, H5, H6, H7, H8, H9, H10, and H12) and seven NA (N1, N2, N3, N4, N6, N8, and N9) subtypes from 16 states (Tables 1, 2). The largest outbreak of LPAI in turkeys occurred in spring and summer 2002 in the Shenandoah Valley in Virginia (VA) and the surrounding states of West Vriginia (WV) and North Carolina (NC). At the time of the outbreak, there were more than 1000 premises and 56 million commercial turkeys and chickens present in the valley. The virus responsible for the outbreak was a LPAI H7N2 virus genetically related to an H7N2 virus known to be present in the LBMS in the northeastern Unites States. Agriculture authorities in VA initially took steps to control the outbreak by diagnostic testing, quarantines, surveillance, and depopulation and disposal of infected poultry. However, the rapid spread and increased number of positive cases quickly overwhelmed the state's capacity to manage the outbreak. Consequently, the Commonwealth of Virginia asked for, and received, assistance from the USDA to control the outbreak. This request was the first time that the Federal Government assumed the responsibility to control an outbreak of LPAI in the United States (control of LPAI is usually managed by individual states) with the subsequent payment of indemnities for destroyed flocks. In total, 197 flocks (4.7 million birds) were destroyed to control the outbreak (1). Of the 197 flocks affected, 153 (72%) were meat turkeys or turkey breeders. The remaining flocks were broilers (13 flocks), broiler breeders (29 flocks), and table-egg layers (two flocks). The outbreak lasted for 4 mo; the last outbreak was diagnosed on July 2, 2002.

In 2002 and 2003, several flocks of commercial meat turkeys in Colorado were infected with subtype H8N4 AIV. The flocks experienced a respiratory disease owing to infection. An inactivated vaccine was developed and used to control the outbreak in turkeys.

In each of 2003, 2004, and 2005, subtype H3N2 was isolated from turkeys in several states; many submissions of serum also were positive for antibodies to the H3N2 AI virus. In each case, the virus was shown to be closely related to H3N2 virus circulating in swine in North America (11; Suarez, pers. comm.).

AI IN DOMESTIC DUCKS AND GEESE

Canada. Following the outbreak of HPAI H7N3 in Canada in 2004, increased surveillance detected LPAI H11N9 in a single duck farm in Ontario. No clinical disease was reported. In 2005, routine surveillance detected an LPAI H5N2 virus in two commercial duck farms in British Columbia located about 2.5 km apart. Both farms (approximately 72,000 ducks and several hundred geese) were depopulated. Wild birds are suspected as the source of the H5N2 virus.

United States. Surveillance activities in the United States detected AI virus or specific antibodies to 10 HA (H1, H2, H3, H4, H5, H6, H7, H9, H10, and H11) and eight NA (N1, N2, N3, N4, N5, N6, N8, and N9) subtypes in domestic ducks and/or geese from 12 states (Tables 1, 2). Some of the infected ducks were raised for release in game hunting reserves, but the majority of the positive ducks were in LBMS in the northeastern United States.

AI IN UPLAND GAME BIRDS

United States. Surveillance for AI infections in upland game birds (e.g., pheasants, quail, chucker partridges) detected virus or antibodies to five HA (H1, H3, H6, H7, and H9) and one NA (N2) subtypes from four states (Tables 1, 2).

AI IN OTHER AVIAN SPECIES

Canada. In 2002, H13N6 was isolated from ring-billed gulls on a lake in Ontario, and in 2003, H3N8 was isolated from imported pet (caged) birds under quarantine in Quebec (Table 1). In 2005, the Canadian Food Inspection Agency reported 15 isolations of AIV from British Columbia (11 H5N2 and four H5N9 isolates), one isolate (H5N1) from Manitoba, one isolate (H5N1) from Ontario, and one isolate (H5N3) from Quebec. The H5 viruses were shown to be LPAI and of North American lineage.

United States. An LPAI H5N2 AI virus was isolated from a (presumably) smuggled 3-mo-old red-lored Amazon parrot in 2002. This isolation is believed to be the first isolation of an H5N2 virus from pet birds (3). Genetic analysis showed the virus to be related to the Mexican lineage of H5N2 viruses. Serum antibodies to subtype H1N1 were found in a commercial pigeon (squab) facility in California in 2003, subtype H2N2 in a loon in Maine in 2005, and subtype H11N2 in a pelican in Florida in 2002 (Table 2). No reports of isolations of AIV from wild waterfowl were available.

AI IN LIVE-BIRD MARKETS

United States. LBMs are commonly found in many parts of the world. The markets serve as a source of poultry meat for local patrons that choose to buy fresh poultry for consumption. In the United States, LBMs have been recognized as a significant reservoir of AI viruses for poultry since 1986, when an epidemiologic link was made between LPAI H5N2 influenza virus infections in commercial poultry in PA and the presence of the same lineage of H5N2 virus in the LBMS in the northeastern United States (7). Since that time, the markets have been routinely monitored for presence of AI virus.

In 1994, an LPAI H7N2 virus was introduced into the northeastern U.S. LBMS; the virus has persisted in the markets despite the efforts by the states to eradicate the virus from the market system. Genetic analysis of isolates recovered over time showed that the H7N2 virus is accumulating mutations (additional basic amino acids near the cleavage site of the H protein) that favor the emergence of a highly pathogenic virus (8). Also since 1996, the lineage of LPAI H7N2 virus found in the LBMS has been linked to at least eight LPAI outbreaks in commercial poultry, resulting in the destruction of millions of birds and an economic loss of millions of dollars to the poultry industry, including costs of eradication and loss of export markets.

Table 3. Prevalence of low pathogenicity H5 and H7 AI in commercial poultry in the United States, 2004[A]. Source: USDA, Animal and Plant Health Inspection Service, Centers for Epidemiology and Animal Health, Fort Collins, CO.

Poultry group	Total production		Positive flocks[B]	% Flock prevalence
	No. birds	No. flocks[A]		
1° Broiler breeders	10,157,252	1058	0	0.0
Broiler breeders	74,198,407	4772	2 LP	0.04
Layer breeder	2,670,074	197	0	0.0
Turkey breeder	5,733,250	696	0	0.0
Commercial broiler	8,492,850,000	223,496	2 LP	0.001
Commercial layer	440,000,000	6774	0	0.0
Commercial turkeys	274,348,000	31,901	0	0.0
Total	9,299,957,083	268,894	4 LP	0.0015

[A]Number of birds tested per flock varies by states; number of flocks tested is not known.

[B]One HPAI flock 6600 chickens for live poultry market; first HPAI since 1984.

From 1994 to 2004, the cleavage site motif of the LPAI H7N2 viruses recovered from the LBMS has changed from a motif with two basic amino acids (PENP**K**T**R**/GLF) to one with four basic amino acids (PE**K**P**KKR**/GLF). However, this change has not resulted in a change in pathogenicity for chickens (IVPI = 0.0). The discovery of isolates with the four basic amino acid motif in January 2002 resulted in the depopulation of all markets in April 2002 followed by cleaning and disinfection and a 3-day rest period (no birds in the markets). Market owners were compensated for the 3-day closure. The H7N2 genotype with the four basic amino acids at the H cleavage site has since been isolated sporadically through December 2005. Studies with this virus by using reverse genetics have shown that H7 viruses probably require an insertional event to become highly pathogenic, unlike the H5 viruses that can become highly pathogenic by mutation or insertions (6).

In each of 2002 to 2005, between 5000 and 9000 specimens from LBMs in northeastern United States were tested by virus isolation for avian influenza virus. From these specimens, 2623 isolations of LPAI H7N2 virus in total were made. Other H7 and H5 LPAI viruses also were detected each of the 4 yr. The subtype and number of isolates (in parentheses) are as follows: 2002, H5N2 (6) and H5N4 (1); 2003, H5N8 (4) and H5N9 (1); 2004, H5N8 (1) and H7N3 (2); and 2005 H5N2 (1) and H5N5 (1). Details on isolations from the LBMs are published each year in the Proceedings of the U.S. Animal Health Association in the report of the Transmissible Diseases of Poultry and Other Avian Species Committee (12).

For several years, the USDA in cooperation with the states and stakeholders has been working to develop a program to eliminate H5 and H7 AI viruses from the marketing system. In 2004, a Uniform Standards document was developed that outlines minimum requirements for each segment of the system—the production units, the distributors, and the LBMs. The document outlines the roles of the state and federal entities, records keeping, licensure, collection of specimens, testing requirements, approved laboratories, and diagnostic tests.

DISCUSSION

The AI outbreaks in North and South America pale in comparison with those caused by the HPAI H5N1 outbreaks reported in Asia, Europe, the Middle East, and Africa since 2003. However, there have been several AI events in the Americas since 2002 that have significantly contributed to our knowledge and understanding of H5 and H7 AI viruses and in the reporting of infections in poultry with these subtypes.

First, following the outbreak in Chile where the HPAI H7N3 virus did not meet the molecular definition for classification as HPAI, the OIE appointed an *ad hoc* committee to rewrite the code chapter on HPAI to reflect inconsistencies with the Chilean H7N3 virus. The revised chapter, which received final approval in May 2005, defines notifiable AI as all H5 and H7 infections in poultry or any AI virus subtype with an IVPI of 1.2 or greater (13). Infections are defined by isolation of the virus, detection of specific RNA, or detection of specific antibodies not related to vaccination.

Second, the viruses responsible for the outbreaks of HPAI in the Americas were unique in different ways. The HPAI outbreaks in Chile and Canada were caused by H7 viruses that mutated to HPAI through a novel mechanism—recombination, involving the nucleoprotein and matrix genes, respectively. These reports were the first reports of recombination associated with increased virulence of AI viruses.

Third, the HPAI H5N2 virus isolated in Texas is the only highly pathogenic virus reported to the OIE where the virus was not pathogenic for chickens. This finding emphasizes the need to do molecular pathotyping of H5 and H7 viruses in addition to the chicken pathogenicity tests to ensure accurate assessment of the virulence potential. The Texas/04, the Chilean/02, and the Canadian/04 viruses all exemplify the lack of predictability of AI viruses and emphasize the need to continually review the criteria used to classify H5 and H7 virus as HPAI as our understanding of these viruses becomes more clear. The economic consequences of reporting a HPAI can be disastrous to a poultry industry, and it is important that reporting criteria accurately reflect appropriate trade risks.

In recent years, the number of HPAI outbreaks has been increasing worldwide. If the HPAI H5N1 outbreak (since 2003) is considered as a single outbreak, there have been 11 HPAI outbreaks reported worldwide since 1995. This number almost equals the 14 HPAI outbreaks reported for the previous 40 yr. There is no single reason for the increase in the number of HPAI outbreaks, but the increasing densities of poultry production worldwide and increased popularity of free-range rearing of poultry probably have contributed to the increased reports of AI in poultry.

From 2002 to 2005, HA subtypes H1 through H13 and all nine NA subtypes were detected in North and South America, consistent with previous reports of the subtypes from wild bird surveys in North America (9). Subtypes H14, H15, and H16 have not yet been detected in the Americas. Control efforts should focus on separation of domestic poultry from wild aquatic birds to reduce the likelihood of introduction of AI viruses into poultry operations.

Finally, the disproportionate number of reports from the United States compared with other countries in North and South America would suggest that AI seems to be common in poultry. However, this is not the case. A study done in 2004, as part of a risk assessment study, showed that infections in commercial poultry with H5 and H7 subtype viruses are very low (0.0015%) considering the size of the poultry industry in the United States (Table 3). Detections of AI in poultry more likely reflect the high level of active and passive surveillance being conducted in commercial and smallholder farms. In each of the 4 yr (2002–05), more than 1.5 million tests were done in the United States by type-specific tests such as the agar gel immunodiffusion or enzyme-linked immunosorbent assays at state and industry laboratories to detect infections in poultry. The number of birds tested per flock varies by state. All positive serum samples and AI virus isolates are submitted for subtyping and characterization to the National Veterinary Services Laboratories, Ames, IA. Therefore, it is likely that the level of testing and reporting are significant factors that may explain the large

number of reports of AI virus infections in the United States and not from other countries.

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